CLINICO-RADIOLOGICAL PROFILE, RISK FACTOR ASSESSMENT, ETIOLOGY AND PROGNOSIS IN CVA

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CERTIFICATE

This is to certify that this is a bonafide work titled **"CLINICO-RADIOLOGICAL PROFILE, RISK FACTOR ASSESSMENT, ETIOLOGY AND PROGNOSIS IN CVA"** is the original dissertation work done by **Dr. L. ANISH** in the Department of General Medicine, Government Stanley Hospital, Stanley Medical College, Chennai – 600 001, in partial fulfillment of the requirements for MD (General Medicine) Branch I Examination, of the Tamil Nadu Dr. M.G.R Medical University, Chennai to be held in March 2009.

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DECLARATION

I **Dr. L. ANISH**, solemnly declare that the dissertation titled **"CLINICO-RADIOLOGICAL PROFILE, RISK FACTOR ASSESSMENT, ETIOLOGY AND PROGNOSIS IN CVA"** was done by me during 2007-2008 under guidance and supervision of Unit chief **Prof. S. MAGESH KUMAR MD.**

This dissertation is submitted to the Tamil Nadu Dr. M.G.R. University towards the partial fulfillment of requirement for the award of M.D. Degree (Branch-I) in General Medicine.

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CONTENTS

INTRODUCTION

World Health Organisation defines the clinical syndrome of "stroke" as 'rapidly developing clinical signs of focal (or global) disturbance of cerebral function with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than vascular origin'. After coronary heart disease (CHD) and cancer of all types, stroke is the third commonest cause of death worldwide. According to Stephen Macmahon et al, Worldwide, about 20 million people suffer from stroke each year; 5 million will die as a consequence and 15 million will survive; of those who survive, 5 million will be disabled by their stroke. A disease of such impact on human mortality and morbidity, stroke continues to take a heavy toll on the productivity of the population and the health care systems.

Time and again it has been proved than stroke is the eventual result of a series of insults on the cerebral and cardiovascular systems. These risk factors not only determine when a stroke will occur, but also the type and severity of the cerebrovascular accident. The risk factors which lead on to stroke differs among different communities, changes with age, sex and many number of reasons. It implies that proper elucidation of the risk factors of stroke and the knowledge of the contribution each risk factor provides to the ultimate culmination in stroke can

help us devise preventive strategies. Since most of the studies are done in populations from developed nations, it is imperative that we need more studies from developing nations like India to find out corresponding statistics.

Even among Indians there are considerable life style differences among populations from various communities and regions. So to be more specific we need studies from south India itself if we have to affirmatively assess the stroke indices in our population.

The etiology of stroke in majority of cases could be identified by a proper history taking, an adequate general examination and judicious use of investigations. A cardiovascular examination and Imaging of the brain usually gives ample information regarding the etiology, type, site and severity of the stroke. Many studies conducted before show that ischemic strokes are common than hemorrhagic and the etiology is different for both. The etiology and site of stroke varies with different communities too. Statistic data on the etiology and site of strokes with proper correlation with brain imagery is still scanty for south Indian population. By this study an attempt is made to find out these indices with respect to a population catered by a tertiary care health system in Urban Tamil nadu.

The clinical features that are common to stroke also varies with type of stroke. For example a history of loss of consciousness and seizures is more in favor of a hemorrhagic lesion. In addition lesions in various sites give rise to

different clinical pictures. An attempt is made to find out the clinical profile of stroke in this study population. The ultimate prognosis of the study population also will be seen. These data will help us to see how they differ from the western data. Thus they will let us set up our own guidelines for stroke management.

AIMS OF THE STUDY

- To find out the various types and sites of stroke in patients admitted in Stanley medical college.
- Find out the clinical profile of stroke in the study population.
- Find out the most common etiologies of strokes in the study group.
- To ascertain the frequency of various established risk factors for stroke.

METHODOLOGY

Exclusion criteria:

*Neurological deficits secondary to epilepsy

* Head injury

* Infective etiology

* Metastatic etiology

* Pre-existing severe physical or cognitive disability.

Inclusion criteria:

Consecutive 100 cases which presented to Stanley Medical College with H/o and examination findings consistent with stroke.

Study period: September 2007 to September 2008

Study Tool:

This is an observational study conducted in a tertiary care set up. Patients fully evaluated and worked up in Stanley medical college, Chennai.

After taking a verbal consent from the patients/relatives, a detailed history is taken and a thorough physical examination (including cardiovascular and neurological) are performed according to a self-designed stroke questionnaire. The questionnaire documents the patient's name, age, sex, past history of transient ischemic attack, valvular heart disease, smoking, alcoholism and family history etc. The findings of the clinical exam are also recorded in this predesigned form which includes the neurological status and associated cardiovascular abnormalities. Carotid bruits, valvular murmurs, Pulse and Blood pressure abnormalities are all noted.

The clinical presentation is observed and it includes general symptoms like loss of consciousness, vomiting, seizures etc and specific ones like hemiplegia, facio brachial monoplegia, cranial nerve palsies, Dysarthria, dysphagia, dysphasias etc. They are classified according to the sex of the patient and the type of stroke subsequently.

The patients are subjected to radiological confirmation by CT scan (all patients) and in selected cases MRI scans and cerebral angiograms as necessary. In hemorrhage cases when needed MR angiograms were taken and suspected cortical venous thrombosis cases were subjected to MR venograms.

The type of stroke, site, extend of lesion, associated anomalies are all noted. Patients are given treatment as needed along with neurology consultations. These patients are subjected to full Quota of blood tests including Complete blood count, renal function tests, liver function tests, Lipid Profiles, Fasting and Post prandial sugar, urinalysis, Chest X rays, ECGs and Echocardiogram in all cases. Coagulation profile was done to rule out coagulopathies in suspected cases.

These patients are further examined daily for 1 week. Their prognosis is noted by the clinical recovery status and by using the Glasgow Coma Scale.

The data is analyzed and sorted to see the types of stroke, and the differences between both sexes. The site is localized clinically and confirmed with CT scan. The various clinical manifestations throughout the hundred cases are categorised. The various risk factors are plotted down partly from history and partly from examination and investigations. History reveals smoking, alcoholism, past rheumatic heart disease, etc. Examination might reveal a carotid bruit or a cardiac murmur. Investigations are done for lipid abnormalities, diabetes and cardiac disorders.

The stoke types- ischemic and hemorrhagic are classified according to sex. Then the site of the lesion is found out with CT scan and strokes are classified according to the Oxfordshire Classification Scale[7] as Total Anterior circulatory

stroke, Partial Anterior circulatory stroke, Lacunar strokes and Posterior circulation strokes. The aetiology is then laid out according to the TOAST classification into Large artery atherosclerosis, Cardioembolism, Lacunar (small vessel disease), other (rare causes, e.g. Vasculitis, Dissection, Fibromuscular dysplasia, Migraine, Venous infarction, other) and Undetermined. In patients with hemorrhage coagulation workup and Platelet counts are done to rule out thrombocytopenic conditions (like leukemias) and hemophilic conditions.

In summary, this thesis is an observational study conducted on patients with clinical features of stroke, confirmed by brain imaging. The study group will be worked up for the etiological diagnosis with full cardiovascular evaluation and follow up for 1 week. The risk factors leading to the stroke and the resultant type, site and prognosis of stroke are studied. This study would thus shed light on the clinico-radiological profile and risk factors underlying stroke in south Indian population.

Definition of Stroke

Stroke is defined as "focal (or at times global) neurological impairment of sudden onset, and lasting more than 24 hours (or leading to death) with no apparent cause other than vascular origin" –WHO[1]

The 24-hour time limit for the duration of symptoms was defined by a WHO committee in 1978 on purely arbitrary grounds.

The definition includes stroke due to:

- Ischaemic stroke or cerebral infarction: inadequate blood supply to a part of the brain as a result of low blood flow, thrombosis or embolism associated with diseases of the blood vessels, heart or blood.
- Haemorrhagic stroke: spontaneous haemorrhage into or over the brain substance, primary intracerebral haemorrhage or subarachnoid haemorrhage respectively.

The definition does not include stroke caused by trauma, infection or tumour that lead to subarachnoid haemorrhage, subdural haemorrhage, epidural haemorrhage, intracerebral haemorrhage or infarction. The definition also excludes subarachnoid haemorrhage with headache, but without a focal or global neurological dysfunction (Hankey & Lees 2001).

Transient ischaemic attack (TIA) which shares common risk factors and pathophysiology to stroke, but is of short duration (defined as symptoms lasting less than 24 hrs) and its outcome does not lead to any disability, as the stroke.

Stroke Syndromes by anatomy[8]	
Cortical strokes	
Middle cerebral artery	➢ Contralateral hemiparesis and sensory loss face and upper extremity more involved. ➢ Contralateral hemianopia ➢ aphasia/aprosodia ➢ gaze abnormalities ➢ extinction on simultaneous touching, apraxia
Anterior cerebral artery	➢ contralateral hemiparesis and sensory loss, lower extremity more involved ➢ disconnection syndrome ➢ abulia, akinetic mutism
Posterior cerebral artery	➢ contralateral hemianopia with macular sparing ➢ disconnection syndrome
Lacunar syndromes of cerebral hemisphere (no diplopia)	
Ventral posterior thalamus	➢ pure sensory loss without weakness
Post limb Internal Capsule	➢ pure motor weakness without sensory loss, confusion or visual field defect
Genu of internal capsule	➢ Dysarthria clumsy hand syndrome
Subthalamic nucleus	➢ Contralateral Hemiballismus
Midbrain syndromes (III nerve palsy or vertical gaze problem)	
Tegmentum, red nucleus, III n	➢ Claude's syndrome: Ipsilateral III palsy & contralateral ataxia
above plus cerebral peduncle	➢ Benedikt's syndrome: above plus contralateral weakness
III nerve + cerebral peduncle (red nucleus spared)	➢ Weber's syndrome: ipsilateral III palsy + contralateral weakness
Dorsal midbrain	➢ Perinaud's syndrome: paralysis of up gaze, convergence- retraction nystagmus, lid retraction
Above + paramedian midbrain	➢ Nothnagel's syndrome: III palsy, vertical gaze paralysis, ipsilateral ataxia
Pontine syndromes (VI nerve, horizontal gaze problem or VII nerve palsy)	
Middle cerebellar peduncle + corticospinal tract	Raymond-Cestan syndrome: ipsilateral ataxia + contralateral weakness
Paramedian pons	One-and-a-half syndrome: ipsilateral horizontal gaze palsy plus contralateral INO
Ventral pons	Millard-Gubler syndrome: VI & VII palsy, contralateral hemiparesis
Medulla syndromes (facial sensory loss\ Horner's syndrome, ipsilateral tongue, palate)	
Dorsolateral medulla	Wallenberg's syndrome: ipsilateral ataxia, Horner's syndrome, facial sensory loss, contralateral loss of pain & temperature
Lateral medulla	Ipsilateral ataxia, horner's syndrome, facial sensory loss, ipsilateral paralysis of soft palate, vocal cords or sternocleidomastoid

Clinical Classification and Aetiological Categorization of Stroke:

The common clinical stroke syndromes are defined using the Oxfordshire classification (Bamford et al. 1991)[7]:

- Total anterior circulation syndrome (TACS) – approximately 20% of cases
- Partial anterior circulation syndrome (PACS) – approximately 30% of cases
- Lacunar Syndrome (LACS) – approximately 25% of cases
- Posterior circulation syndrome (POCS) – approximately 25% of cases

These syndromes are based on the clinical features (Table 1). However, they can be further refined by CT or MRI brain scan findings. Classification of the stroke syndromes is important as it provides valuable information about the anatomical and vascular location, aetiology and prognosis of the stroke (Table 2) (Hankey & Lees 2001).

Ischaemic stroke can be further divided into various subtypes, as defined by the TOAST study, the Trial of Org 10172 in Acute Stroke Treatment (Adams et al. 1993) (Table 3). This subtype classification is closely associated with the underlying pathophysiology of ischaemic stroke and can be used in targeting treatment.

Table 1: Stroke Syndrome Using Oxfordshire Classification Scale[7]

Classification	Syndrome
Total Anterior Circulation syndrome (TACS)	Combination of: ➢ Hemiparesis+/-hemisensory loss ➢ Homonymous hemianopia ➢ Global aphasia (dominant hemisphere) or visuo-spatial deficit/neglect (non-dominant hemisphere) with or without drowsiness
Partial Anterior Circulation Syndrome (PACS)	Two of the three components of TACI Dysphasia typically related to either expressive or receptive. Typically no drowsiness
Lacunar Syndrome (LACS)	➢ Pure motor stroke, or ➢ Sensory-motor stroke, or ➢ Pure sensory stroke, or ➢ Ataxic hemiparesis, or ➢ Dysarthria clumsy-hand syndrome No hemianopia or cortical dysfunction
Posterior Circulation Syndrome (POCS)	Ipsilateral cranial nerve palsy with contralateral motor and/or sensory deficit/neglect Conjugate gaze disorder Cerebellar dysfunction without ipsilateral long tract signs Isolated homonymous hemianopia

Source: Adapted from Hankey 2001 (Hankey & Lees 2001).

Table 2: Anatomy, Pathology, Aetiology and Prognosis of the Four Clinical Stroke Syndromes

	TACS	PACS	LACS	POCS
Anatomy	Fronto-temporalparietal lobes or thalamus/internal capsule/occipital	Lobar	Small deep lesion in corona radiata, internal capsule, thalamus or ventral pons	
Pathology, (%)				
Infarction	85	85	95-98	85
Hemorrhage	15	15	2-5	15
Aetiology				
Infarction	Occlusion of ipsilateral ICA or MCA, and occasionally PCA; by embolism from heart, aortic arch or carotid or vertebrobasillar arteries, or insitu thrombosis	Occlusion of branch of MCA or PCA; by embolism from heart, aortic	Usually lipohyalinosis, microatheroma or 'complex' disease (fibrinoid necrosis) of small perforating artery. Rarely arteritis or embolism	Occlusion of VBA or PCA, or branches; by in-situ thrombosis or embolism from heart, aortic arch or VBA
Haemorrhage	Any of possible causes	Any of possible causes	Any of possible causes	Any of possible causes
Recurrence rates	Low	High in first 3 months	Low but steady over 12 months	High first 2 months and steady over 12 months

TACS, Total Anterior Circulation Syndrome; PACS, Partial Anterior Circulation Syndrome; LACS, Lacunar Syndrome; POCS, Posterior Circulation Syndrome; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; VBA, vertebral-basilar artery.

Source: adapted from Hankey 2001 (Hankey & Lees 2001).

Table 3: TOAST Classification of Aetiology of Ischaemic Stroke

Aetiology of Ischemic Stroke

1. Large artery atherosclerosis
2. Cardioembolism
3. Lacunar (small vessel disease)
4. Other (rare causes, e.g. Vasculitis, Dissection, Fibromuscular dysplasia, Migraine, Venous infarction, other)
5. Undetermined

TOAST, Trial of Org 10172 in Acute Stroke Treatment

Source: Modified from Adams (Adams et al. 1993).

Epidemiology of Stroke

Mortality

According to the World Health Organisation (WHO), stroke is the second worldwide leading cause of mortality. In addition a large proportion of the population is debilitated and rendered functionally dependent. Worldwide, about 20 million people suffer from stroke each year; 5.5 million will die as a consequence and 14.5 million will survive; of those, who survive, 5 million will be disabled by their stroke. Of those deaths, two thirds occur among people living in developing countries. So, the global burden of stroke needs to be defined for developed and developing nations.

Morbidity

In terms of disability, stroke is among the five most important causes of disability in both developing and developed countries (World Health Organisation). It will lead on to long term disability and a burden on health care facilities.

The Global Burden of Disease (GBD)[3] Study, in 1990, reported 9.4 million deaths in India of which 61,900 were from “Stroke” and the disability adjusted life years (DALYs) lost almost amounted to 28.5 million - nearly six times higher than that due to malaria. When these estimates were projected for the year 2020, Murrey

and Lopez[4] reported that 61 million DALYs are likely to be lost due to stroke, of these 52 million (84%) will be in the developing countries. Reddy and Yusuf[9] have reemphasized the "Health Care and Economic Consequences" of emerging epidemic of cardiovascular diseases in developing countries.

Incidence and Prevalence

The incidence and prevalence of stroke varies in different communities and countries. For example, it is estimated that approximately 4.5 million Americans are currently living with the effects of stroke, and that every year another 570,000 will survive a stroke that results in disability. Stroke is the third most common cause of death in the United States.[2] According to WHO the age-standardised incidence rates per 100,000 varied from 101 to 285 in men and from 47 to 198 in women. (Thorvaldsen et al. 1995)[6]

Brain stroke is the third largest killer in India after heart attack and cancer and the second largest in the world, according to the World Health Organisation (WHO). India will face an enormous socio-economic burden to meet the costs of rehabilitation of "stroke victims" because the population comprising the peak years (age 55-65) of occurrence of stroke is gradually increasing. However, for stroke prevention planning, reliable epidemiological information on pattern of disease and

exposure to risk factors and morbidity or mortality trends for CVD in defined populations is not available. Recent community surveys[15] for "hemiplegia" presumed to be CVD, identified 320 cases in 145,456 persons, indicating an overall Crude Prevalence Rate (CPR) of 220 per 100,000 persons.

During the last decade, the age-adjusted prevalence rate of stroke was between 250-350/100,000. Recent studies showed that the age-adjusted annual incidence rate was 105/100,000 in the urban community of Kolkata and 262/100,000 in a rural community of Bengal. The ratio of cerebral infarct to hemorrhage was 2.21. Hypertension was the most important risk factor. Stroke represented 1.2% of total deaths in India.

Many studies have shown that the incidence and mortality of stroke has declined in the past 20 years, particularly in many developed countries (Sudlow & Warlow[11] 1996; Rothwell *et al.* 2004)[12]. Two prospective stroke studies,[14] during the period 1963-1968 and 1978-1982 in Bombay, using identical methodologies, it was observed that there was a significant drop in case fatality rate (32% to 12%) thereby resulting in a higher survival (68% to 88%) but with residual disability. Thus, these changing trends have posed a major social challenge in occupational rehabilitation and in solving the needs for stroke survivors. These data suggest that India is already facing "Stroke Epidemic".

Regional and Community wise variations

Incidence and risk factors of stroke vary among different communities and regional groups. For example, in the US, the incidence of cerebrovascular disease is higher in African Americans and some Hispanic Americans (Howard et al. 1994; Gorelick 1998)[10]. Understanding the ethnic variations of stroke epidemiology may be of value in defining stroke prevention and management strategies; especially in multiethnic populations. The incidence of stroke is heterogeneous among populations and changes over time. A large number of studies have been conducted to examine stroke incidence and prevalence as well as stroke types across different regions and countries.

In Indian subcontinent with the multitude of regional and cultural variations, studies need to be conducted in every subset to identify the distribution and risk factors of stroke. This will enable us in targeting effective therapy in a customized manner. For example, a recent survey on 20,842 rural residents in East India[13] reported a crude prevalence rate for stroke in elderly (age 41-60 yrs) at 540/100,000, which differs in statistics from the rest of the country. This thesis intends throw light into this situation regarding the South Indian scenario.

Comparison of Stroke Type in various regions

Reference	Country		Sample size	Ischaemic stroke (%)	Haemorrhagic stroke (%)	SAH (%)	Unknown (%)
(Sze *et al.* 2000)	HK, China		793	84	16	--	--
(Giroud *et al.* 1989)	Dijon, France		203	80	15	5	5
(Saposnik & Del Brutto 2003)	South America		--	51-73	26 - 46	--	--
(Saposnik *et al.* 2000)	Argentina, South America		361	73	27	--	--
(Hajat *et al.* 2001)	South London,UK	Whites	995	81	13	6	--
		Blacks	230	69	19	12	
(Johansson *et al.* 2000)	Sweden		1318	74	11.5	4.5	10
(Bamford *et al.* 1988)	Oxfordshire, UK		675	81	10	5	5
(Lefkovits *et al.* 1992)	Melbourne, Australia		925	75	16	9	o

Stroke Risk Factors and Risk Factor Management

Overview of Stroke Risk Factors

Although stroke mortality is declining in the west, identifying the clinical patterns and the risk factors and intervening to control or modify them remain the most important means of reducing stroke incidence[16]. Most of the local, South Asian and the far eastern studies have suggested that the proportion of intracerebral hemorrhage was significantly higher 21% to 45% than in the west 10% to 20% while cerebral infarction varied between 55% to 70.1% in the local studies and 60% to 84% in the western.[16, 17, 18] Although some determinants of stroke, such as age, gender, race, ethnicity and heredity cannot be modified, they are risk markers.

Identification and management of stroke risk factors is an important strategy for both primary and secondary stroke prevention. Risk factors for stroke have been classified as non-modifiable, partially modifiable and modifiable (Goldstein *et al.*2001). There are also potential risk factors that have been described in a number of recent publications (Sacco 2001).

The significance of a risk factor for an individual is determined by the relative risk RR), which represents the strength of the association between the risk factor and the chance of having a stroke (Hennekens & Buring 1987). On the other hand, the population attributable risk (PAR) is more relevant to the importance of a risk factor in a population (Hennekens & Buring 1987). For example, although the

relative risk of hypertension for stroke is not high, the fact that the prevalence of hypertension in the population is high means that there is a high population-attributable risk of hypertension. Therefore, treatment of hypertension in the general population has a higher impact on the prevention of stroke than that of a risk factor with a lower prevalence but higher relative risk, e.g. atrial fibrillation.

Summary of Risk Factors for Ischaemic Stroke[19]

Ischaemic stroke	**Relative risk**	**Estimated Age standardized prevalence of exposure in population (%)**	**Population attributable Risk (%)**
Non-modifiable			
Age	--	--	--
Gender	--	--	--
Heredity(race, ethnicity, family history)	--	--	--
Modifiable – well documented			
Hypertension	2-4	30	15-22.5
Diabetes Mellitus	2	20-25	12.5
Atrial Fibrillation	6	1	0.8
Lipid disorder (hypercholesterolemia)	1.8-2.6	51	25
Cigarette smoking	2-4	25	12.5 – 18.8
Peripheral vascular disease	1-4	3	0 – 2.25
TIA or previous stroke	7	2	1.7
Ischaemic heart disease (IHD)	1-3	20	0 – 13.3
Carotid bruit/stenosis	3-15	4	0.4 – 0.9
Modifiable – less well-documented			
Obesity	--	60	--
Heavy alcohol drinking	--	10	--
Illicit drug use	--	--	--
Physical inactivity	--	54	--
Poor nutrition	--	--	--
Oral contraceptives	--	--	--
Migraine	--	--	--
Hemostatic and inflammatory factors	--	--	--
Homocysteine	--	--	--
C-creative protein	--	--	--

Summary of Risk Factors for Haemorrhagic Stroke[20]

Major risk factors
Hypertensive small vessel disease (fibrinoid necrosis)
Vascular malformations
Intracerebral tumours
Septic arteritis and mycotic aneurysms
Hypertension, isolated systolic blood pressure
Left ventricular hypertrophy
Prior stroke
Amyloid angiopathy
Anticoagulation, thrombolytic therapy
Clotting factor deficiency
Leukaemia and thrombocytopenia

Minor risk factors
Heavy alcohol use
Cocaine and other sympathomimetic drugs
Age
Socioeconomic status
Very low serum cholesterol
Cigarette smoking

Non-Modifiable Risk Factors

Age, gender, family history, race and ethnicity are non-modifiable risk factors for stroke (Goldstein *et al.* 2001)[21]. These factors denote the population group that will be at a high risk for stroke, but cannot be targeted by preventive measures.

Age

In adults, stroke risk more than doubles in each decade over age 55 years (Wolf *et al.* 1992). The mean age of stroke presentation 57 to 71 years is relatively lesser in Asian population than in the west 76 to 80 years.[19, 22, 23.] Increasing age is clearly the strongest determinant of the number of new cases of stroke each year. (Simons *et al.* 1998)[24]; 97% of ischaemic stroke occur among people over the age of 45 (Adams *et al.* 1992)[25]. About 75% of strokes occur in people 65 years and older and approximately 50% occur in people aged greater than 75 years (Bamford *et al.* 1990)[7]. The cause of stroke also varies by age. In the elderly, atherosclerosis and cardio embolism secondary to atrial fibrillation are the leading causes of ischaemic stroke (Atrial Fibrillation Investigators 1994), while cerebral amyloid angiopathy and chronic hypertension are leading causes of intracerebral haemorrhage in the elderly (Rasool *et al.* 2004)[26].

Family History

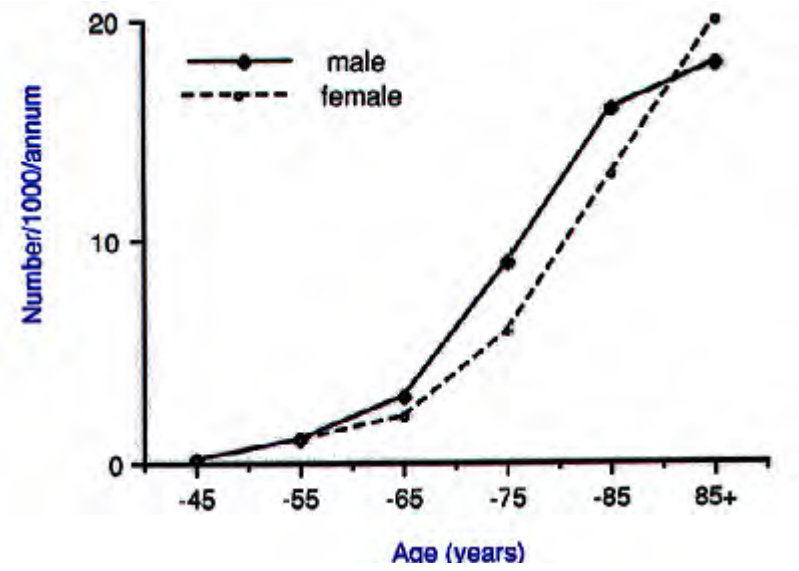
Persons with a family history of stroke are put to an increased risk for sustaining a stroke. It may be due to sharing of common cultural patterns and lifestyle or due to inherited coagulopathies or tendencies for intracranial aneurysms and AV malformations. The risk of stroke increases by 40 and 140% respectively with a maternal or parental history of stroke or TIA.[27]

Gender

Stroke risk factors, type and aetiology differ between men and women. Men are at a greater risk for stroke than women. Women tend to live longer than men who die of other co morbidities; as a result, they often outnumber men in stroke prevalence figures.[19]

Age-specific incidence rate of stroke for men within the 45 to 84 yr age group is observed to be 174/100,000 against 122/100,000 in females.[19]. This 30% higher incidence of stroke in men has been observed in many studies. (Bamford et al. 1988)[7]. Gender has a role in the cause of stroke also. Strokes due to pregnancy, puerperium, oral contraceptive use, migraine or saccular aneurysms are more common in women, while atherosclerosis is more common in men (Adams et al. 2002).[25]

Incidence of Stroke in Male and Female versus Age, Results from Oxfordshire7 Study



Race and Ethnicity

After coronary heart disease (CHD) and cancer of all types, stroke is the third commonest cause of death worldwide. Compared to Caucasians, Asians have a lower rate of CHD and a higher prevalence of stroke[28.] Among the Asians, the number who died from stroke was more than three times that for CHD.[29,30] In one report, the age standardized, gender-specific stroke mortality rate was 44 to 102.6/100,000 for Asian males, compared with only 19.3 for Australian white males.[31] In the early 1980s the prevalence rates of stroke were around 500-700 per 100,000 in the western countries and 900 per 100,000 in Asia.[32] The disparity between the stroke and CHD incidence is usually attributed to high prevalence of

hypertension and low levels of blood lipids among Asians.[33] Hypertension was related to high salt intake and perhaps to genetic factors and low serum lipid was due to low levels of animal fats and protein in oriental diet. Among Asians stroke has been found to be more common among the Chinese and Japanese. The incidence of stroke for African American is more than double that of Caucasians (233/100,000 versus 93/100,000)[19, 34] Again it appears to be influenced by the prevalence of certain risk factors in that particular race. The African Americans have a high prevalence of hypertension, sickle cell disease, poor diet and lower economic and social factors than Caucasians.[21]

Modifiable Risk Factors

According to the American Heart Association (AHA), modifiable risk factors are defined as "well documented", "less well-documented" or "potential" risk factors (Goldstein *et al.* 2001). [21] The major risk factors are as follows.

Hypertension

Hypertension is the single most powerful and important modifiable risk factor causing stroke. In hypertensives a fourfold greater risk of stroke at systolic blood pressure values ≥160 has been observed.[19] Treatment of hypertension reduces stroke risk by approximately 38%. [21] Reduction of blood pressure is associated with a reduced incidence of stroke in elderly by 40% (Sacco et al. 1997)[19]. The

most prevalent form of hypertension among elderly is Isolated Systolic hypertension. The risk of stroke increases with systolic blood pressure in all age groups. However, the effects of blood pressure lowering agents may vary in different age groups (Prospective Studies Collaboration 1995).[20]

Diabetes Mellitus

Diabetes mellitus is an important risk factor for stroke. It causes chronic end organ damage, accelerates atherosclerosis and leads on to stroke and heart disease. The risk of stroke in patients with diabetes mellitus is about 4 times that found in normal individuals.[19] Poorly controlled diabetes mellitus is associated with long-term damage, dysfunction and failure of various organs; including eyes, kidneys, nerves, heart and blood vessels (Meneilly & Tessier 2001).[25] It is an important risk factor for heart disease and stroke.[19, 36] The frequency of diabetes mellitus was found to be higher in Indian population 18%-42.5% [17] than in the western 10% to 26%. [37]

Cigarette Smoking and tobacco chewing

Cigarette smoking is considered to be a major risk factor for stroke. The estimated relative risk for stroke among smokers is 1.5 to 2.9 times that of nonsmokers (Shinton & Beevers 1989).[40] Many studies have confirmed the role of active cigarette smoking as a major risk factor for stroke.[38, 39] Smoking leads on to increased atherosclerosis and vessel wall changes. Smoking causes reduced

blood vessel distensibility and compliance by leading to increased arterial wall stiffness. Smoking causes increased fibrinogen levels, increased platelet aggregation, decreased high-density lipoprotein (HDL) levels and increased haematocrit (Goldstein et al. 2001).[21]

The relative risk of ischaemic stroke in current smokers has been estimated as 1.9 However, cessation of smoking reduced the risk of stroke and returned the relative risk to the baseline level after 5 years of cessation (Goldstein et al. 2001)[21], which makes it an important intervention in the prevention of stroke. After 5–10 years, people who quit smoking reduce their risk of stroke to that of nonsmokers.[19]

Many local studies have shown somewhat similar pattern of smoking/tobacco chewing as those in the west.[37, 41, 19, 22, 23] Betel nut chewing with or without tobacco is associated with increased risk of stroke according to some studies.[45] Areca nut chewing will lead on to increased periodontal disease which in turn can lead on to increased stroke.[43, 44] For example, the results of a meta-analysis of nine cohort studies indicate that the relative risks of future cardiovascular events and stroke for persons with periodontal disease were 1.44 and 2.85, respectively. [42] Further studies are needed to confirm and to find out the reasons why cerebrovascular mortality is increased in tobacco chewing.

Alcohol Drinking

The protective effect of moderate alcohol drinking has been demonstrated in a number of epidemiological studies. [19, 46]In the Melbourne Risk Factor Study long term heavy alcohol consumption was identified as a major risk factor. Heavy alcohol consumption has been associated with an increased incidence of haemorrhagic stroke (Thrift et al. 1999)[46]. According to another study drinking habits were associated only with deaths from ischemic stroke, and the risk patterns were different for men and women.[47] Heavy drinking may be considered when males take more than 3 drinks (33-45g) and females take more than 2 drinks (22-30g) per day.

Atrial Fibrillation

Atrial fibrillation is an important risk factor for stroke. In the Copenhagen stroke study it was shown that Stroke in patients with AF is generally more severe and outcome markedly poorer than in patients with sinus rhythm. This accentuates the importance of anticoagulant treatment of individuals with AF[48] Its prevalence begins to increase in both genders after age 40 years and rises rapidly after age 65 years. AF increased steeply with age in the stroke population, from 2% in patients <50 years old, 15% in patients in their 70s, and 28% in patients in their 80s, to 40% in patients ≥90 years of age.[48] It is also an important attributing factor for stroke outcomes (Marini et al. 2005).[49] AF is a common cause and independent

risk factor for cardio-embolic stroke. It is associated with about 16% of all ischaemic stroke (Wolf et al. 1991a).[50] Studies like the Stroke Prevention in Atrial Fibrillation (SPAF) III trial showed that anticoagulation therapy using warfarin is superior to aspirin in the prevention of cardio-embolic strokes in individuals with high risk of stroke (Hart *et al.* 2003). AF is a predictive factor for severe stroke and early death in acute ischaemic stroke.[53]

Lipid Disorder

Lipid profiles, including total blood cholesterol, High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL) and triglyceride levels have been used as indicators for the risk of stroke. Increased level of total cholesterol, especially LDL, is associated with a higher incidence of ischaemic stroke.[51] But a low level of total cholesterol can be associated with an increased risk of haemorrhagic stroke.[46, 51] Over the past few years it has been demonstrated that strong correlation between lipid lowering therapy and the incidence of stroke exists. A meta-analysis showed that statins reduced the incidence of fatal and non-fatal stroke by 20-25% (Briel et al. 2004).[52]

Hyperhomocysteinemia

Homocysteinemia is a stroke risk factor.[19] It predisposes young individuals for strokes and other vascular events due to the procoagulant state it creates. It is a sulphur-containing amino acid derived from catabolism of the

essential amino acid methionine. Abnormalities in the function of certain enzymes that metabolise methionine, or deficiencies in folic acid, vitamin B6, or vitamin B12 cofactors, can lead to elevated homocysteine levels – called Hyperhomocysteinemia. Endothelial injury and acceleration of atherosclerosis is noted in hyperhomocystinemia. The increased risk of stroke in Hyperhomocysteinemia is contributed by both genetic and environmental factors. A meta-analysis of 30 observational studies showed that a 25% lower usual homocysteine level was associated with a 19% lower stroke risk (Homocysteine Studies Collaboration 2002).

Physical activity

The Framingham study results indicate that medium and high levels of physical activity among men are protective against stroke relative to low levels.[54] The protective effect of physical activity in the elderly was observed in the Northern Manhattan Stroke study. In women physical activity, including moderate-intensity exercise such as walking, is associated with substantial reduction in risk of total and ischemic stroke in a dose-response manner.[55]

Comparison of stroke risk factor profile												
Reference	Country	Sample size	HT	DM	AF	IHD	MI	PVD	TIA	Lipid	Smoking	CHF
(McGruder *etal.*	US	Whites 1603 (71%)	62	21	--	34	24	--	--	--	20	--
		Blacks 438 (19%)	79	32	--	29	21	--	--	--	26	--
		Hispanics 224 (10%)	70	38	--	30	24	--	--	--	13	--
(Harvey *et al.*	Chicago US	830	76	34	17	32	--	6	--	--	53	11
(Hajat *et al.* 2001)	South London UK	Total 1254	58	17	22	25	--	--	17	--	70	--
		Whites 995 (79%)	56	14	25	27	--	--	18	--	74	--
		Blacks 203 (16%)	71	34	7	16	--	--	15	--	52	--
(Burn *et al*1994)	Oxfordshire UK	545	47	11	18	24	--	12	18	--	12	--
(Jorgensen *et al.*1994)	Copenhagen Denmar	500	33	16	16	22	10	16	9	--	45	--
(Sze *et al.* 2000)	Hongkong	Chinese 793	57	30	10	6	1	1	2	12	20	2
HT, hypertension; DM, diabetes mellitus; AF, atrial fibrillation; IHD, ischaemic heart disease; MI, myocardial infarction; PVD, peripheral vascular disease; TIA, transient ischaemic attack; CHF, congestive												

Stroke Prevention

Stroke once occurs can lead on to mortality and significant morbidity. Since our knowledge regarding the etiology and risk factors of stroke is increasing day by day, it is evident that strokes can be prevented. The various risk factors of stroke that can be modified should be targeted. It would be prudent to identify high risk groups and stroke survivors and provide stroke preventive therapy to them. Successful prevention eliminates the expenses of acute stroke treatment, rehabilitation and subsequent lost productivity and long-term care and disability cost (Hankey 1999). This involves treating conditions like diabetes, dyslipidemia and hypertension and also life style modification strategies like smoking and alcohol cessation, physical activity and dietary modifications.

Risk factor modification can be attempted either by targeting the general population or the high risk populations.[19] Attempting to provide stroke prevention therapy to the general population is not cost effective. Whereas identifying and giving therapy to people with risk factors for stroke is cost effective and leads to a significant reduction in morbidity and mortality from stroke. There are primary and secondary preventive strategies for stroke. A patient with symptomatic cerebrovascular disease is likely to have other cardiovascular diseases or is predisposed to develop them. Preventive measures should complement reduction in

risk of atherothrombotic events in the coronary arteries and other arterial territories.

Primary Prevention of Stroke

Primary prevention is aimed at preventing an individual's first ever stroke. This involves preventing the risk factors from emerging as well as treating them. So individuals with no risk factors like hyper tension and diabetes are advised regarding measures to prevent them from occurring. This includes smoking and alcohol cessation, taking on healthy diet and exercise patterns and so on. This involves the use of mass media and public education systems.[19] Stroke prevention strategies deployed after development of well-established risk factors for stroke have developed are also having a significant reduction in stroke incidence. For example, lowering blood pressure and anticoagulant therapy for atrial fibrillation have reduced the incidence of stroke significantly (Atrial Fibrillation Investigators 1994; O'Brien *et al.* 1999; Donnan *et al.* 2003b). So by targeting such population a heavy economic benefit is obtained by reducing the number of strokes and the DALY s which would have been lost otherwise.

Secondary Prevention of Stroke

Secondary prevention is given to prevent recurrent stroke in stroke survivors. This also includes treatment of people who have evidence of atherosclerosis such as myocardial infarction, angina pectoris, claudication, amaurosis fugax and transient

ischaemic attack (Adams *et al.* 2002). The risk of recurrent stroke can still be reduced by risk factor modification (Organizing Committees 1998). In developing nations many of the time patients are picked up and risk factors identified only after they sustain a stroke. So, secondary prevention of stroke holds the key to reduce the morbidity and mortality in such high risk populations.

Therapies for Prevention of Stroke

Several interventions have been identified as lowering the incidence of stroke. They are enumerated below.

Antihypertensives:

Treating and controlling hypertension is one of the most effective strategies to reduce the incidence of stroke. The successful management of hypertension is probably one of the leading reasons why stroke incidence has declined during the past 20 years. (Hall *et al.* 1991, the HOPE study). Several studies show that no demonstrable floor exists for the relation between blood pressure and risk of stroke, with risk continuing to halve for every 10 mm Hg fall in diastolic pressure even at conventionally normotensive values.[57] Meta-analyses of small trials of antihypertensive treatment in patients with a stroke showed a 28% reduction in relative risk for stroke regardless of baseline blood pressure.[56] This was supported by the post-stroke antihypertensive study (PATS),[58] in which

indapamide gave an absolute reduction in stroke risk by 2.9% compared with placebo over three years in 5665 patients with prior stroke or transient ischaemic attack and mean baseline blood pressure of 154/93 mm Hg.

The rate of stroke in patients with hypertension is age dependent. Results from a pooled analysis from 45 prospective studies involving 450,000 patients showed that the absolute stroke rates were 2, 5 and 8 per 1000 for age group <45, 45-65 and >65 respectively (Prospective Studies Collaboration 1995). The Heart Outcomes Prevention Evaluation (***HOPE***) study was a randomised, placebo-controlled trial in patients with a history of vascular disease, with 4645 patients randomised to receive 10mg once a day of ramipril and 4652 patients to receive a placebo. Results showed that there was a reduction in stroke incidence in the intervention group (Relative Risk Reduction, RRR = 32%, Absolute Risk Reduction, ARR = 1.5%), In the Perindopril Protection Against Recurrent Stroke Study (***PROGRESS***), a combination of perindopril and indapamide significantly reduced the risk of recurrent stroke.

Antithrombotic Therapy:

Antithrombotic therapy includes antiplatelet (e.g. aspirin, clopidogrel, and dipyridamole) and anticoagulation (e.g. warfarin) therapy.

In a meta-analysis of results of 21 randomized trials comparing antiplatelet therapy with placebo in 18 270 patients with prior stroke or TIA,

antiplatelet therapy was associated with a 28% relative odds reduction in nonfatal strokes and a 16% reduction in fatal strokes.[61] Antiplatelet usage after transient ischaemic attack also has been shown to be effective in stroke prevention (Sacco & Elkind 2000; Antithrombotic Trialists' Collaboration 2002).

The effectiveness and safety of antiplatelet therapy for primary stroke prevention in patients with atrial fibrillation has been examined in many large-scale trials. Many studies have established an irrefutable role for aspirin in preventing stroke. These studies include the Stroke Prevention in Atrial Fibrillation (SPAF I) study, the Atrial Fibrillation, Aspirin, Anticoagulation (AFASAK I) study and the Low-dose Aspirin, Stroke, Atrial Fibrillation (LASAF) study. A total of 1965 subjects were included in these three trials with a mean follow-up period of 1.3 years (equivalent to 2560 patient years). The results showed that aspirin was associated with a reduction of ischaemic stroke and all cause mortality.

The efficacy of clopidogrel was compared with that of aspirin in the Clopidogrel Versus Aspirin in Patients at Risk of Ischemic Events (CAPRIE) trial. It showed that efficacy of clopidogrel in the dose of 75 mg daily is comparable to Aspirin 325 mg daily. It was associated with lesser gastrointestinal symptoms.

The meta-analysis by Hart et al using adjusted dose warfarin with a total of 2900 subjects, found that warfarin reduced stroke by 62% with an absolute risk

reduction of 2.7% per year for primary stroke prevention and 8.4% per year for secondary stroke prevention. The Stroke Prevention in Reversible Ischemia Trial (SPIRIT) was stopped early because of increased bleeding among those treated with high-intensity oral anticoagulation (INR, 3.0 to 4.5) compared with aspirin (30 mg/d) in 1316 patients. For patients with non cardioembolic ischemic stroke or TIA, antiplatelet agents rather than oral anticoagulation are recommended to reduce the risk of recurrent stroke and other cardiovascular events. But anticoagulant therapy is more potent than antiplatelet for stroke prevention in patients with atrial fibrillation.

Preventing embolism:

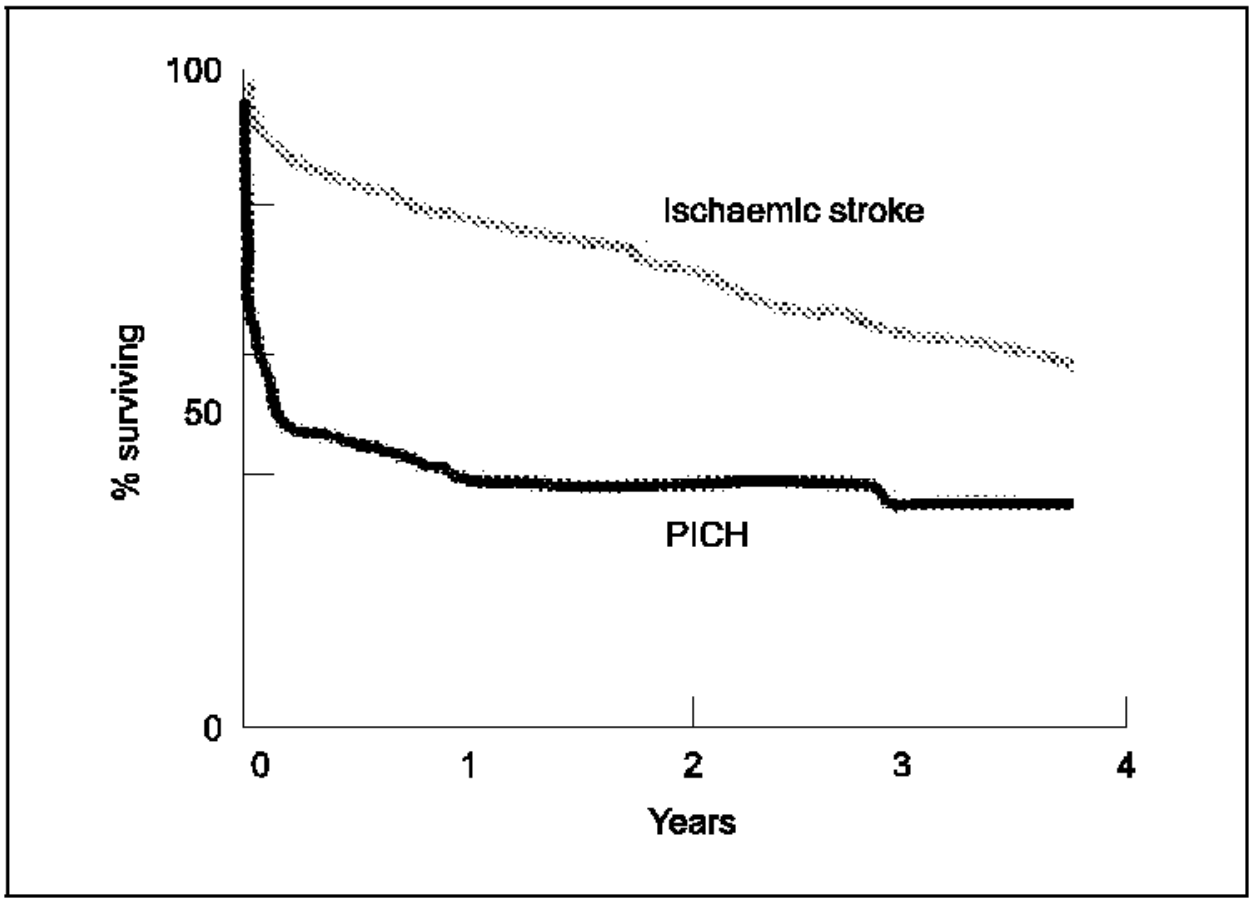
For patients with atrial fibrillation, dose-adjusted warfarin sodium is administered to keep the international normalized ratio [INR] in the 2 to 3 range; target 2.5) unless there is a specific contraindication for that medication.[59] In the latter case, the patient should be treated with aspirin 50 to 325 mg/d.

In patients with TIA or mild stroke and symptoms referable to severe (70% to 99%) carotid artery stenosis (or to moderate [50% to 69%] stenosis in a patient with significant risk factors), the treatment of choice is carotid endarterectomy by a surgeon with a low complication rate (morbidity and mortality ,6%).[60] For patients with TIA or mild stroke who do not have atrial fibrillation or

moderate-to-severe carotid stenosis, treatment with a daily dose of 50 to 325 mg of aspirin is of demonstrated benefit.

Stroke Prognosis

Several factors affect the prognosis after stroke. They include the age of the person, the size, aetiology of stroke and so on. Both short and long term mortality is associated with stroke. Studies have shown that the 30-day mortality for ischaemic stroke is approximately 8-20% (Sacco 1997)[19] versus 40-45% for haemorrhagic strokes (Dennis 2003)[63]. However, despite this initial significant difference, the long-term prognosis has been reported to be similar independent of the stroke type, although the mortality rate remains higher for haemorrhagic than for ischaemic stroke (Dennis 2003)[63]The figure shows the survival rate over four years from the Oxfordshire7 Community Stroke Project, comparing ischaemic and haemorrhagic strokes (Dennis et al. 1993)[62]



PICH, Primary Intracerebral Haemorrhage. *Source: (Dennis et al. 1993)*[62]

Stroke Outcome Measures

Stroke outcome is measured for short term as well as long term. Outcomes have been described using a number of measurements, but have mainly been categorised according to mortality, disability and quality of life. Outcome measures have also been categorised into three different domains – survival rate, disability / functional status and quality of life / psychosocial consequence (Flick 1999). There is no consensus concerning the measures that best reflect stroke related disability. Survival rate has been determined according to stroke severity and stroke subtypes. Most common impairment measures are NIH stroke scale and

Modified Mathew scale. Activity measures include Barthel Index and Rankin Handicap Scale. There are several other methods to assess stroke outcome. Patients' emotional, cognitive and living arrangements have also been used as indication of the psychosocial consequences. The most common Outcome measure in rehabilitation is Functional Independence Measure which incorporates motor, social and cognitive performance.

General data

Total number of patients:	100
Number of males:	64
Number of females:	36
Number of Infarcts:	88
Number of hemorrhages:	12
Number of males with infarct:	48
Number of females with infarct:	40
Number of males with hemorrhage:	8
Number of females with hemorrhage:	4

Clinical features

General:		**Infarct**			**Hemorrhage**	
		Total	**Male**	**Female**	**Male**	**Female**
Loss of consciousness:		58	32	15	7	4
GCS	11-15	25	14	11	0	0
	5-10	34	17	15	2	0
	<5	41	18	13	6	4
Seizures:		7	1	0	4	2
Urinary incontinence:		36	15	11	7	3
Vomiting:		16	3	5	6	2
Headache:		44	15	19	8	2
Hemiplegia\ Hemiparesis:		72	40	21	7	4
Facio brachial Monoplegia:		16	10	6	0	0
Monoplegia:		5	3	2	0	0
Crossed hemiplegia:		3	1	2	0	0
Hemianesthesia:		18	11	7	0	0
Hemianopia:		11	7	4	0	0
Pure cerebellar ataxia:		2	1	1	0	0
Ataxic Hemiparesis:		4	3	1	0	0
Aphasia:		29	16	11	1	1
Cranial nerves excluding Cr7:		4	3	1	1	0

Anatomical sites of stroke with sex distribution

	Male	Female
TACS: Total	7	4
Infarct:	5	3
Hemorrhage:	2	1
PACS: Total	24	19
Infarct:	20	17
Hemorrhage:	4	2
LACS: Total:	17	14
Infarct:	16	14
Hemorrhage:	1	0
POCS: Total:	8	7
Infarct:	7	6
Hemorrhage:	1	1
Total	56	44

Aetiology

Infarct:	Total 88	Male 48	Female 40
Cardioembolism:	9	3	6
Large Artery Atherosclerosis:	17	10	7
Lacunar stroke (small vessel disease):	51	29	22
Other:	3	1	2
Undetermined:	8	5	3

Hemorrhage:

Primary Intracerebral hemorrhage with or without SAH:

Total: 10

Male: 7

Female: 3

Primary SAH:

Total: 2

Male: 2

Female: 0

Intracranial Hemorrhage sites:

Subcortical	4
Thalamic	2
Capsuloganglionic	2
Cerebellar	1
Brainstem	1

Risk factors

Non-modifiable

Age and Gender:

Age	Total		Infarct		Hemorrhage	
	male	female	Male	female	male	female
15-24	0	1	0	1	0	0
25-34	0	1	0	1	0	0
35-44	4	4	4	3	0	1
45-54	22	13	20	12	2	1
55-64	27	11	26	11	1	0
65-74	7	5	4	3	3	2
75-84	4	1	2	1	2	0

Modifiable				**Infarct**		**Hemorrhage**	
	Total	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
Hypertension:	52	28	24	22	20	6	4
Diabetes mellitus:	34	20	14	19	14	1	0
Dyslipidemia:	14	8	6	8	5	0	1
Cigarette smoking:	36	34	2	29	2	5	0
Ischemic heart disease:	18	13	5	12	4	1	1
Carotid bruit/Stenosis:	3	2	1	2	1	0	0
Atrial fibrillation:	9	3	6	3	6	0	0
TIA or previous stroke:	8	5	3	5	3	0	0
Peripheral vascular disease:	2	2	0	2	0	0	0
Obesity:	7	5	2	5	2	0	0
Heavy Alcohol drinking:	12	11	1	6	0	5	1
Illicit drug use:	2	2	0	2	0	0	0
Physical inactivity:	9	6	3	6	3	0	0
LVH:	18	12	6	6	3	6	3
Vascular malformations:	2	2	0	0	0	2	0

Prognosis

	Improved	Deteriorated	Dead
total	69	25	6
males	49	11	4
females	20	14	2

RESULTS

A total of 100 patients were enrolled in the study after leaving out those who didn't satisfy the inclusion criteria. Out of the 100 stroke patients 64 were males and 36 were females.

Out of this population 88 patients had ischemic stroke. It included 48 males and 40 females. Hemorrhagic stroke was seen in 12 patients. It included 8 males and 4 females.

Males considerably outnumber females with stroke in this study, both in ischemic and hemorrhagic strokes. 88% of the people had ischemic stroke when compared to 12% of hemorrhagic strokes.

Among the clinical features studied, it was seen that most patients presented with hemiparesis/hemiplegia. It amounted to 72 % of the cases. Males accounted for 47 cases and females 25. That amounts to 65.3 % of male cases and 34.7% of females. Loss of consciousness was the next most common feature seen in 58%. These findings were the most noted clinical features in both sexes. Next in the order of frequency came headache (44%) and then urinary incontinence (36%). Next only to them came specific findings like aphasia (29%) and Hemianesthesia (18%).

53.4 % of infarct patients had loss of consciousness while 91.7% of patients sustaining hemorrhagic stroke had loss of consciousness. Less than 2 % of infarct cases presented with seizures while 50% of hemorrhage patients had seizures. 29.55% of patients with ischemic strokes had urinary incontinence while 83.33% of patients with hemorrhagic strokes suffered urinary incontinence. When 66.67% of hemorrhagic stroke patients had vomiting, only 9% of ischemic stroke patients had vomiting. 69% of ischemic stroke patients had hemiparesis/ hemiplegias, while 92% of the hemorrhagic stroke patients sustained unilateral weakness.

Other features like Hemianesthesia, crossed hemiplegias, aphasias, cerebellar features, and ataxic hemiparesis all were common in ischemic infarcts.

Majority of the patients presented with a grave GCS score <5 at admission (41%). 34% of patients had GCS between 5 and 10 and 25% had a GCS between 11 and 15. 83% of the hemorrhagic strokes had a GCS less than 5. All females who had hemorrhagic stroke had GCS less than 5 but only 75% of males with hemorrhage had GCS less than 5. Favorable GCS scores (11-15) were observed only in ischemic strokes.

On analyzing the anatomic location of cerebral lesions it is seen that the majority of cases are Partial Anterior Circulatory Stroke amounting to 43% of the total. Ischemic stroke in the Partial Anterior Circulatory stroke category

predominates with a total of 37%. Ischemic strokes prevail high in all the circulatory areas and males have an increased prevalence in all areas. Second in the order are Lacunar ischemic strokes with 30% of the total. Posterior ischemic strokes come third with 13% of total. Among hemorrhagic strokes Partial anterior hemorrhagic strokes top the list with 6% of the total. Total anterior circulatory strokes were the ones least observed in this study. Another thing which is noted is that only 1% of the patients had lacunar hemorrhage, while lacunar infarcts were observed in 30 % of the patients.

Regarding the etiology of ischemic stroke the maximum numbers of cases were due to small vessel disease rather than large vessel arthrosclerosis. These small vessel diseases were identified by CT evidence of lacunar infarcts and by ruling out atherosclerosis in larger vessels. They constitute 51% of the total. Large artery atherosclerosis producing widespread brain damage and generalized atherosclerotic evidence like carotid bruits were found in 17% of total strokes. A cardiac source of embolus could be found in 9 patients of which 7 had Rheumatic heart disease with atrial fibrillation and 2 had ischemic dilated cardiomyopathy. The category “others” include 3 cases of ischemic stroke caused by cortical venous infarcts. There were 2 females and 1 male. Both female patients were postpartum cases but the male had APLA positive status. In spite of extensive evaluation 8

cases remained elusive of a diagnosis and are categorised and undetermined which included 5 males and 3 females.

Males predominate in lacunar and atherosclerotic disease, while this study shows that in this population females have an increased incidence of cardioembolic stroke and cortical venous thrombosis. One fold increased incidence of Cardioembolism noted in this study may be due to the increased prevalence of Rheumatic mitral valve disease in Indian females.

Radiologic correlation shows that among hemorrhage cases in this study out of the total 12 cases, 10 had primary intraparenchymal bleed. None of them were found to have berry aneurysms. The site of maximum predilection was Subcortical area (33%) followed by thalamic and Capsuloganglionic bleeds (16% each). There were cerebellar and brainstem bleeds too. Primary Sub arachnoid bleed was seen in 2 cases; both were males with berry aneurysms.

Analysis of risk factors show that age plays a very important role in stroke. In this study maximum number of cases occurred in the age group of 55-64 years which contributed 38% of the cases. That is followed by the 45- 54 year old group with 35% of cases. When ischemic strokes alone are considered the above said distribution is still followed. But when hemorrhage is considered maximum incidence is found in the 65-74 year group with 5 % of the total strokes being

them. On observing the gender wise distribution, ischemic stroke is seen to occur maximally by 55-64 years for males and earlier by 45-54 years for females. Hemorrhagic stroke in males and females were maximal during 65-74 years. Only 10 cases occurred less than 45 years which included 4 males and 6 females.

On analyzing the risk factors, it is clearly seen that Hypertension stands out as the number one risk factor for both ischemic and hemorrhagic strokes. 52% of the stroke patients had hypertension. Among them 80.7% were ischemic strokes and 19.3 % were hemorrhagic strokes. Hence it will be seen hypertension commonly results in ischemic stroke. 83% of the people with hemorrhagic strokes had hypertension where as only 47.7% of ischemic stroke patients had hypertension.

Next to hypertension, the risk factor maximally found was cigarette\beedi smoking. Even though the sheer total amounted to 36% of the total stroke sufferers, it will be seen that 94% of them were males. Since only 2 female smokers were there in the study, this risk factor predominantly affects males. In fact cigarette smoking was found in 53% of the males who had stroke, where as hypertension was seen in only 43.7% of males had hypertension. Hence even though the total number of stroke patients with hypertension is more than any other risk factor, in this study it is seen that cigarette smoking stays number one among

males. Among females hypertension is the top most risk factor with 66.67% of them having hypertension.

Diabetes mellitus came as the third highest risk factor with 34% of stroke patients having it. It came up to the third maximum risk factor for males and the second maximum risk factor for females. 31% of males with stroke had diabetes, while 38% of females had diabetes mellitus.

Ischemic heart disease was found in 18% of people along with LVH in 18% of stroke patients. 10% of ischemic stroke patients had LVH while 75% of hemorrhagic stroke cases had LVH, corresponding with the increased association with hypertension.

14% of stroke sufferers had dyslipidemia affecting 12.5% of males and 16% of females. Since majority of the female patients were post menopausal, this increased association with dyslipidemia and stroke may imply the influence of postmenopausal hormonal status on the lipid and cardiovascular status. 92.86% of them had ischemic strokes compared to 7.14% of hemorrhagic strokes.

Alcoholism was found in 12% of stroke patients. The only female alcoholic enrolled into the study sustained hemorrhagic stroke. Next to hypertension and LVH, alcoholism contributes maximum as a risk factor to hemorrhagic strokes (50%).

Obesity, peripheral vascular disease, TIA, physical inactivity, atrial fibrillation, carotid bruits, and illicit drug use all were associated with ischemic strokes. 2 patients, both of them males, who had vascular malformations sustained hemorrhagic strokes.

Of the total of 100 cases 6 patients had died by the end of 1st week. Even though 4 males had died when compared to 2 females, it amounted to 6% of total males and 5% of females. Of these cases 3 were hemorrhagic strokes and an equal number of ischemic strokes, which amounted to 25 % of hemorrhagic strokes and 3.4% of ischemic strokes. Thus it can be clearly seen that hemorrhagic strokes had very high mortalities and males and females were equally affected. 25% of the cases deteriorated which included 7 hemorrhagic strokes (58%) and 18 ischemic strokes (20%). Hence morbidity is also more in hemorrhagic strokes. 17% of males had deteriorated and 38.9% of females had deteriorated. 69% of total stroke sufferers improved at the end of one week. 76% of male stroke sufferers improved while only 55 % of females had improvement. Thus in this study it is noted that females had more morbidity and poor recovery even though mortality rates were comparable between both sexes.

DISCUSSION

In this study stroke patients have been observed for a period of 1 week. The clinical features, risk factors, type and site of stroke are all noted along with the week end prognosis. The findings observed are herewith described relating to the conclusions of some other similar studies. Since the differences in profile so brought to light will allow us to make conclusions regarding the community in which this study was done.

In this study a 28% greater incidence of stroke in males (64%) when compared to females (36%) was noted. 30% higher incidence of stroke in men has been observed in many studies. (Bamford et al. 1988)[7]. Strokes due to pregnancy, puerperium, oral contraceptive use, migraine or saccular aneurysms are more common in women, while atherosclerosis is more common in men (Adams et al. 2002).[25]

Ischemic stroke (88%) is much more common than hemorrhagic stroke (12%). This goes well in accordance with American Heart Association's estimate that 87% of all strokes are ischemic, the remainder being intracerebral and subarachnoid hemorrhage strokes

Most common clinical feature was hemiplegia/hemiparesis (72%), followed by loss of consciousness (58%), headache (44%) and urinary incontinence (36%). This is corroborated by other studies done in India like the one by Man Mohan Mehndiratta et al. which found that 80.3% had hemiplegia. Loss of consciousness, seizures, urinary incontinence and vomiting were all common in hemorrhagic stroke when compared to ischemic strokes. Females with hemorrhagic stroke present with lesser GCS than men with hemorrhagic strokes

Partial anterior circulatory strokes were most common (43%). Most common Ischemic strokes were partial anterior circulatory strokes (37%), but so do hemorrhagic strokes (6%) in this territory itself. Similar results were obtained by Qiu F[64], et al. where PACS was seen in 51% and LACS in 34%. In our study LACS was found in 30%. Bamford et al.[7] showed similar results with 25 % of cases with LACS.

Most common etiology was small vessel disease amounting to 51%, followed by large artery atherosclerosis (17%). In both cases males predominate. According to study conducted in china[64] 28.6% of patients were found to have small vessel disease.

Our population is seen to have more of atherosclerosis and small vessel disease when compared to other studies.

Females had one fold increased incidence of cardio-embolism, which may be due to the increased prevalence of Rheumatic heart disease among females.

The age group with maximum incidence is 55-64 years amounting to 38% of total.

The age group 45-64 years together constituted 73% of stroke in this study. Where as in western communities stroke incidence peaks much later. This may be due to our risk factor profile and other genetic constitutions. The smaller life expectancy if Indians may be another reason for earlier peaking of stroke in Indians. In Framingham heart study 64% of cases occurred between 65-84 yrs age group and only 30.15% cases occurred in the 45-64 yrs group. Hemorrhagic strokes occurred much later at 65-74 yrs maximally.

Hypertension, the most common risk factor for both ischemic and hemorrhagic strokes and was found in 52% of patients. Hypertension commonly resulted in more ischemic strokes (80.7%) when compared to hemorrhages. Other studies prove that hypertensives have a fourfold greater risk of stroke at systolic blood pressure values ≥160.[19] Data from other studies also show that treatment of hypertension reduces stroke risk by approximately 38%. [21]

Cigarette smoking was the second greatest risk factor for stroke (36%), but among males it stands as the most common risk factor pushing hypertension to the second place. Honolulu heart programme affirmatively links cigarette smoking

with both ischemic and hemorrhagic strokes. In this study cigarette smoking had more correlation with hemorrhagic strokes.

Diabetes mellitus came as the next greatest risk factor with 34% of stroke patients suffering from it. According to some studies the frequency of diabetes mellitus was found to be higher in Indian population 18%-42.5% (sandercock et al.)[17] than in the western population 10% to 26%. [37] This increased prevalence of diabetes was reflected in this study too.

Ischemic heart disease and LVH was found in 18% of patients each and LVH was more associated with hemorrhagic stroke. This may be due to the fact that in this study that 83% of hemorrhagic stroke patients had hypertension.

Dyslipidemia affected 14% of patients and affected more females than males. It may be due to the postmenopausal hormonal changes resulting in dyslipidemia. Dyslipidemia resulted in ischemic strokes predominantly. Some western data correlate with this fact showing that increased level of total cholesterol, especially LDL, is associated with a higher incidence of ischaemic stroke.[51]

Alcoholism was found in 12% of stroke patients and was more associated with hemorrhagic lesions. Other studies revealing the association between Heavy alcohol consumption and an increased incidence of haemorrhagic stroke include the one done by Thrift et al. 1999[46].

Increased mortality and morbidity is seen with hemorrhagic strokes when compared with ischemic strokes. Even though mortality rates were comparable between both sexes, females had more morbidity and poor recovery in this study. Studies have shown that the short term mortality for ischaemic stroke is approximately 8-20% (Sacco 1997)[19] versus 40-45% for haemorrhagic strokes (Dennis 2003)[63].

CONCLUSIONS

- Stroke is more common in males (64%) when compared to females (36%).
- Ischemic stroke (88%) is much more common than hemorrhagic stroke (12%).
- Most common clinical feature was hemiplegia/hemiparesis (72%), followed by loss of consciousness (58%), headache (44%) and urinary incontinence (36%).
- Loss of consciousness, seizures, urinary incontinence and vomiting were all common in hemorrhagic stroke when compared to ischemic strokes.
- Majority of the patients presented with GCS <5. Among them females had a preponderance of having had a hemorrhagic stroke when compared to males. So females with hemorrhagic stroke present with lesser GCS than men with hemorrhagic strokes. Favorable GCS scores above 11 were observed only in ischemic strokes.
- Partial anterior circulatory strokes were most common (43%).
- Most common Ischemic strokes were partial anterior circulatory strokes (37%), but so do hemorrhagic strokes (6%) in this territory itself.

- Lacunar strokes were more often infarcts (30%) when compared to hemorrhages (1%). They are the most common sites of stroke after partial anterior circulatory strokes.
- Most common etiology was small vessel disease amounting to 51%, followed by large artery atherosclerosis (17%). In both cases males predominate.
- Cardio-embolism was found in 9% and Rheumatic heart disease with atrial fibrillation was the most common cause. Females had one fold increased incidence of cardio-embolism, which may be due to the increased prevalence of Rheumatic heart disease among females.
- Cortical venous thrombosis was more common among females.
- Primary intra-parenchymal bleed was more common than subarachnoid hemorrhages. The site of maximum predilection was subcortical area (33%).
- The age group with maximum incidence is 55-64 years amounting to 38% of total, most of them being ischemic. Where as in western communities stroke incidence peaks much later. This may be due to our risk factor profile and other genetic constitutions.
- Hemorrhagic strokes occurred much later at 65-74 yrs maximally.
- Hypertension, the most common risk factor for both ischemic and hemorrhagic strokes and was found in 52% of patients. Hypertension

commonly resulted in more ischemic strokes (80.7%) when compared to hemorrhages. But considering all those who had hemorrhages brings the conclusion that 83% of them were hypertensives when compared to 47.7% of ischemic stroke patients having had hypertension.

- Cigarette smoking was the second greatest risk factor for stroke (36%), but among males it stands as the most common risk factor pushing hypertension to the second place. But among females hypertension remains the most common risk factor.
- Diabetes mellitus came as the next greatest risk factor with 34% of stroke patients suffering from it.
- Ischemic heart disease and LVH was found in 18% of patients each and LVH was more associated with hemorrhagic stroke. This may be due to the fact that in this study that 83% of hemorrhagic stroke patients had hypertension.
- Dyslipidemia affected 14% of patients and affected more females than males. It may be due to the postmenopausal hormonal changes resulting in dyslipidemia. Dyslipidemia resulted in ischemic strokes predominantly.
- Alcoholism was found in 12% of stroke patients and was more associated with hemorrhagic lesions.

- Increased mortality and morbidity is seen with hemorrhagic strokes when compared with ischemic strokes. Even though mortality rates were comparable between both sexes, females had more morbidity and poor recovery in this study.

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Proforma: CLINICO-RADIOLOGICAL PROFILE, RISK FACTOR ASSESSMENT, ETIOLOGY AND PROGNOSIS IN ACUTE CVA

Name: Age: Sex: MRD No:

Serial No:

COMPLAINTS:

Clinical H/o: Hemiplegia/Hemiparesis:

Past H/o Stroke: Faciobrachial monoplegia:

Past H/o TIA: Monoplegia:

Loss of consciousness: Crossed hemiplegia:

Seizures: Ataxic hemiparesis:

Vomiting: Pure cerebellar ataxia:

Headache: Hemianesthesia:

Urinary incontinence: Hemianopia:

Aphasia: Crania nerves other than facial N:

RISK FACTORS	Yes	No
Systemic HT	☐	☐
Diabetes Mellitus	☐	☐
CAD	☐	☐
Other Heart diseases	☐	☐
Cigarette smoking	☐	☐
Dyslipidemia	☐	☐
Carotid bruit/ stenosis	☐	☐

Atrial fibrillation:	☐	☐
H/o TIA or previous stroke	☐	☐
Peripheral vascular disease	☐	☐
Obesity	☐	☐
Heavy alcohol drinking	☐	☐
Illicit drug use	☐	☐
Physical inactivity	☐	☐
LVH	☐	☐
Vascular malformations	☐	☐

EXAMINATION FINDINGS:

Height: Weight: BMI:

PR: BP:

CVS: **RS:** **GIT:**

CNS: GCS:

Deficit: Power:

Cranial Nerve palsy:

Sensory: Cerebellar:

INVESTIGATIONS

Hb: ESR S. Cholesterol:

FBS: PPBS: TG:

Blood Urea: CXR: LDL:

S. Creatinine: ECG:

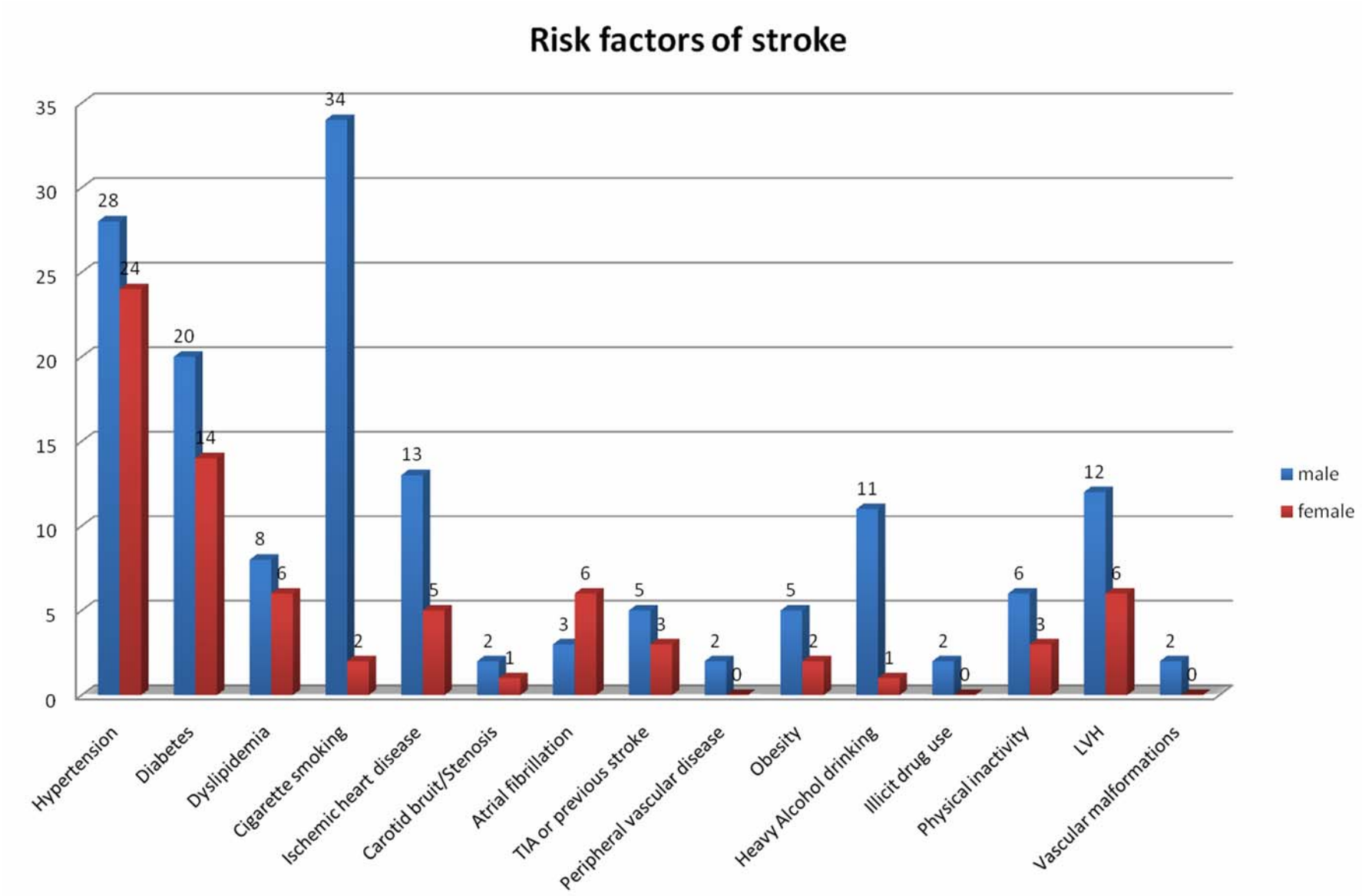
CT scan findings:			**Yes**	**No**
Type:	Infarct		☐	☐
	Hemorrhage	Intra parenchymal	☐	☐
		SAH	☐	☐
	CVT		☐	☐
Location of infarct:		Total Anterior Circulation Stroke	☐	☐
		Partial Anterior Circulation Stroke	☐	☐
		Lacunar Stroke	☐	☐
		Posterior Circulation Stroke	☐	☐
Location of intraparenchymal hemorrhage:		Subcortical	☐	☐
		Thalamic	☐	☐
		Capsuloganglionic	☐	☐
		Cerebellar	☐	☐
		Brainstem	☐	☐
Aetiology of infarcts:		Cardioembolism	☐	☐
		Large Artery Atherosclerosis	☐	☐
		Lacunar stroke (small vessel disease):	☐	☐
		Other:	☐	☐
		Undetermined:	☐	☐

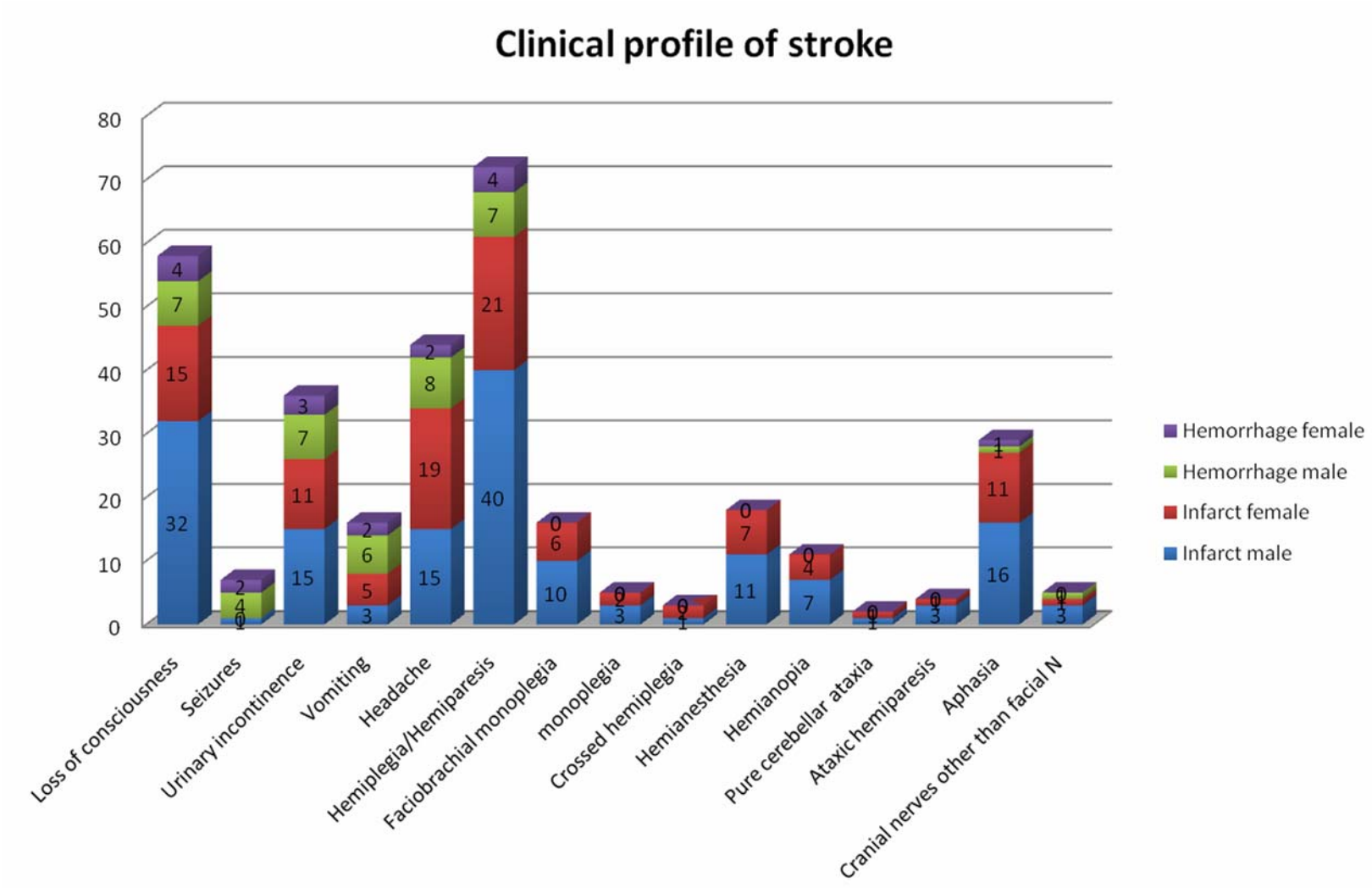
Progress after 1 week:

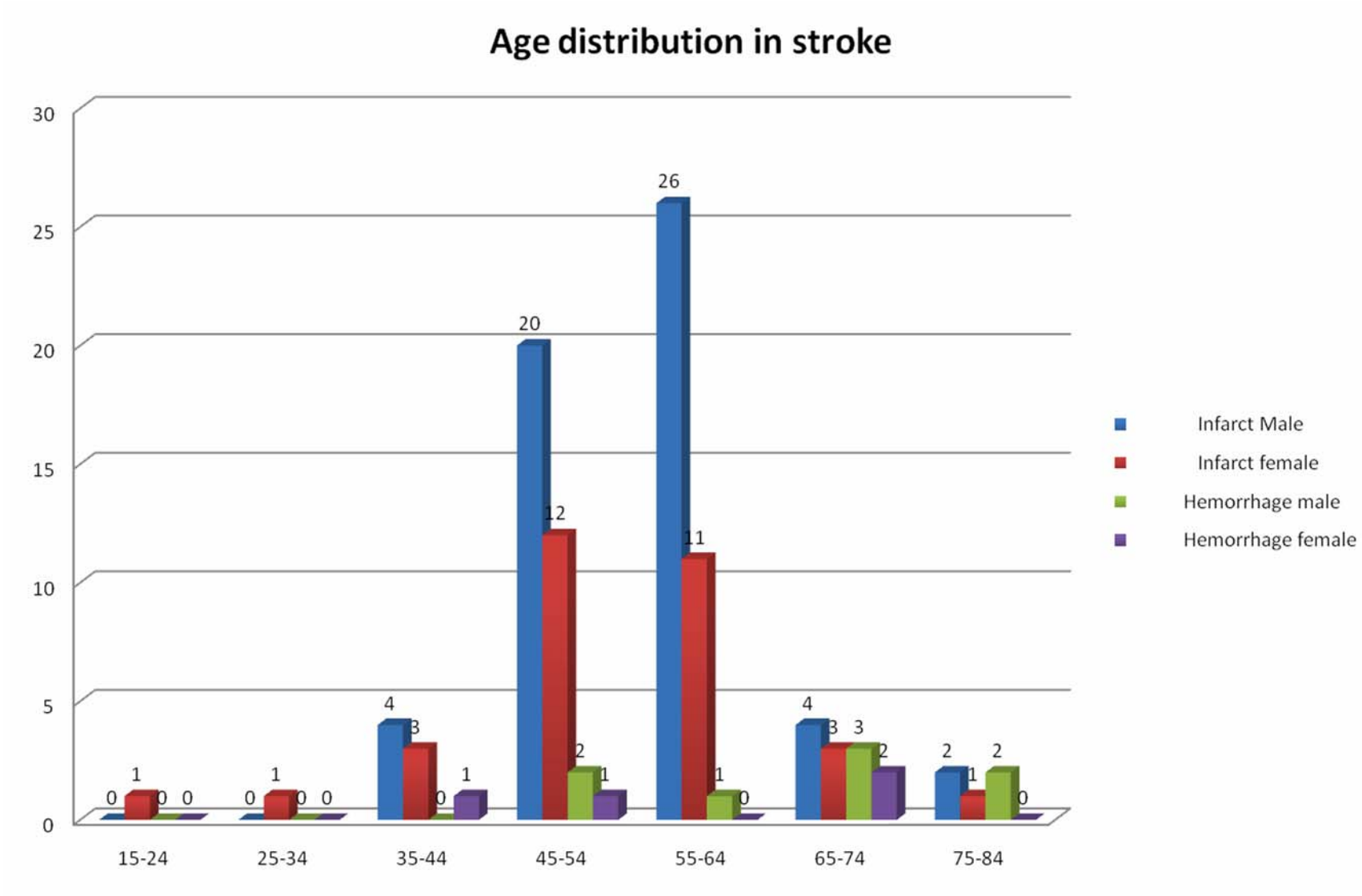
Improved

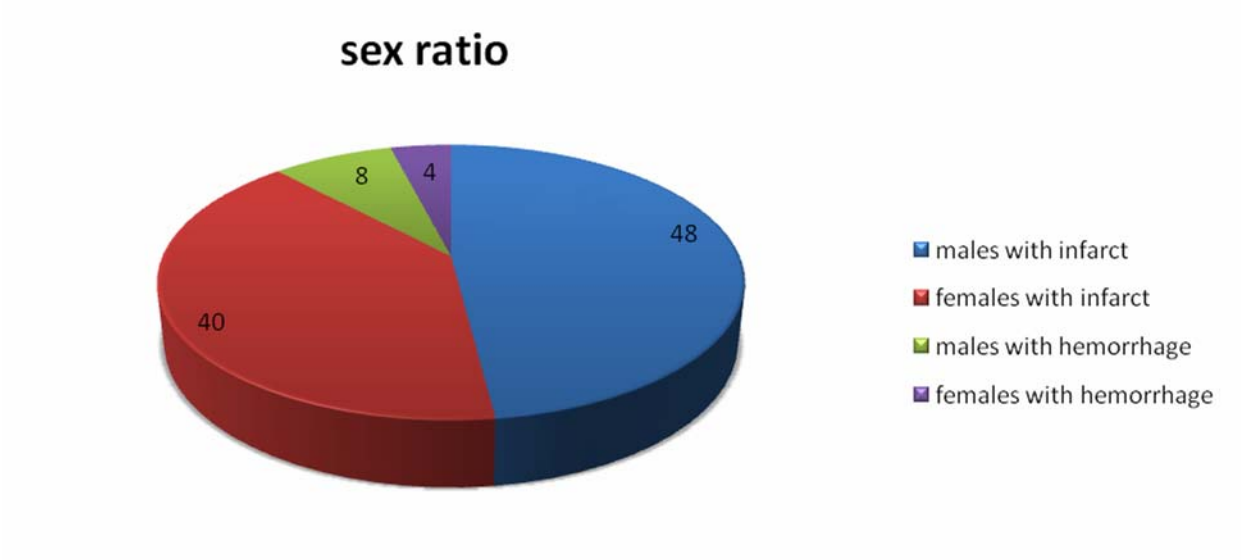
Deteriorated

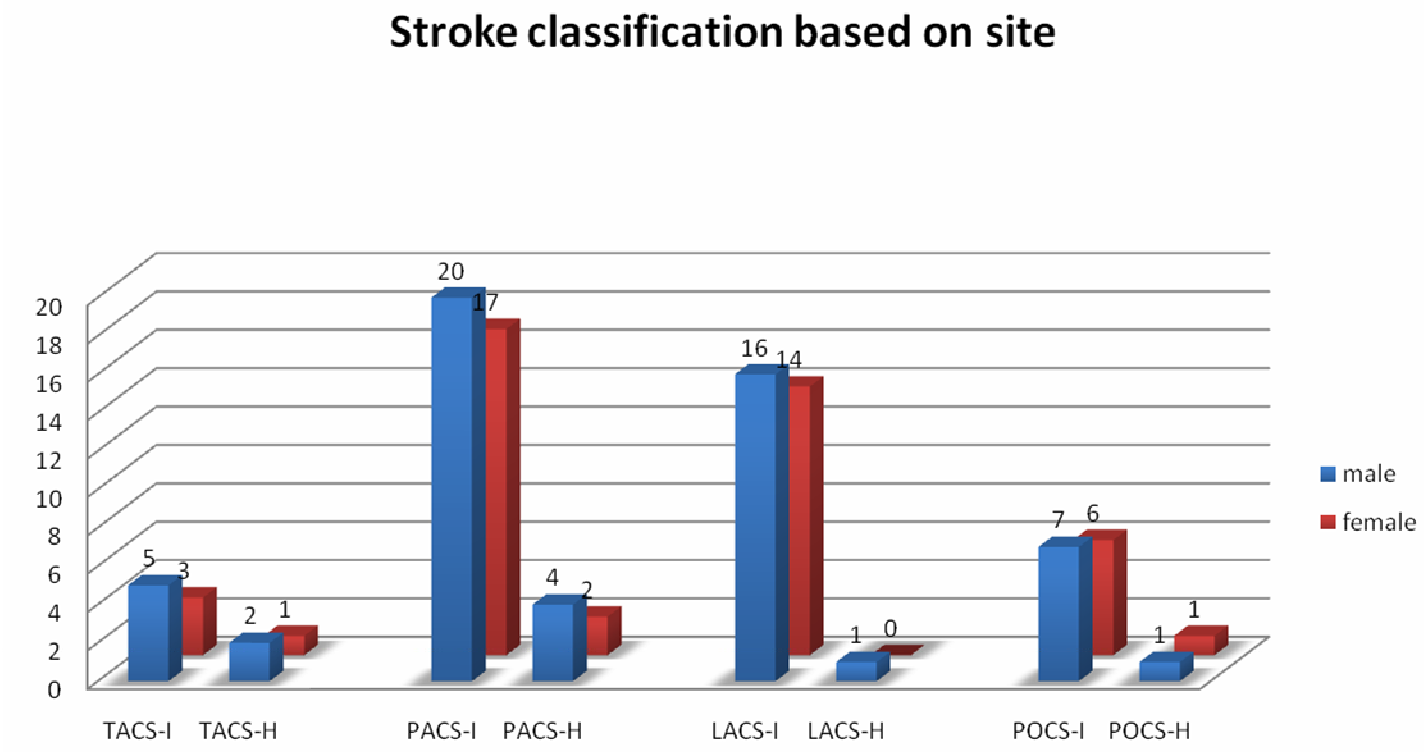
Expired


Risk factors of stroke
35
30
25
20
15
10
5
0
28
24
20
14
8
6
34
2
13
5
2
1
3
6
5
3
2
0
5
2
11
1
2
0
6
3
12
6
2
0
male
female
Hypertension
Diabetes
Dyslipidemia
Cigarette smoking
Ischemic heart disease
Carotid bruit/Stenosis
Atrial fibrillation
TIA or previous stroke
Peripheral vascular disease
Obesity
Heavy Alcohol drinking
Illicit drug use
Physical inactivity
LVH
Vascular malformations


Clinical profile of stroke
80
70
60
50
40
30
20
10
0
Loss of consciousness
Seizures
Urinary incontinence
Vomiting
Headache
Hemiplegia/Hemiparesis
Faciobrachial monoplegia
monoplegia
Crossed hemiplegia
Hemianesthesia
Hemianopia
Pure cerebellar ataxia
Ataxic hemiparesis
Aphasia
Cranial nerves other than facial N
Hemorrhage female
Hemorrhage male
Infarct female
Infarct male

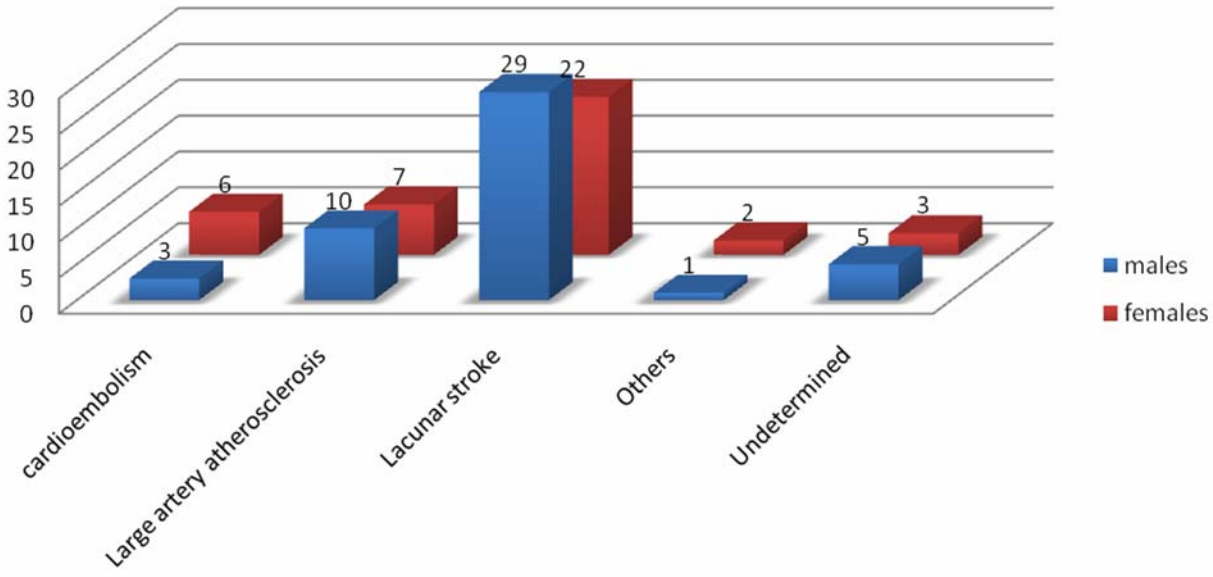

Age distribution in stroke
30
25
20
15
10
5
0
15-24
25-34
35-44
45-54
55-64
65-74
75-84
Infarct Male
Infarct female
Hemorrhage male
Hemorrhage female





TACS- Total Anterior Circulatory Stroke, PACS- Partial Anterior Circulatory Stroke, LACS- Lacunar Stroke, POCS- Posterior Circulatory Stroke
I – Infarct and H- Hemorrhage

Aetiology of infarcts



Intracerebral Hemorrhage sites